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1996 Summer Neuropeptide Conference

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NEUROPEPTIDE Y AND ITS RECEPTORS IN THE REGULATION OF REPRODUCTIVE HORMONE SECRETION. Jon E. Levine, Janice H. Urban, Angela C. Bauer-Dantoin, Leslie M. Besecke, and Sarah L. Leupen. Department of Neurobiology and Physiology, Northwestern University, Evanston, IL 60208.

Neuropeptide Y (NPY) is released at several levels of the reproductive axis to regulate female reproductive cyclicity. In the hypothalamus, NPY regulates release of luteinizing hormone-releasing hormone (LHRH) into the hypophysial portal vasculature; at anterior pituitary gland, NPY exerts pronounced modulatory effects on LHRH-stimulated LH secretion. In a series of studies, we have examined the integral role that NPY secretion may play at these loci in the production of mid-cycle gonadotropin surges. Using *in situ* hybridization of NPY mRNA in hypothalamus, we have documented a clear-cut increase in NPY gene expression that is specifically associated with the elaboration of LH surges in proestrus rats. That NPY release is augmented in concert with this increased NPY expression has been confirmed. In addition to an acute increase in NPY expression in anticipation of LH surges, we have also characterized an acute up-regulation of both hypothalamic (LHRH release) and pituitary (LHRH-induced LH release) responsiveness to the facilitory effects of NPY. Pharmacological analysis revealed that a Y1 or Y1-like subtype mediates NPY's actions. We therefore performed quantitative RT-PCR assays of Y1 receptor mRNA throughout the estrous cycle of female rats, to determine if increased NPY receptor expression mediates increased tissue responsiveness to the peptide. While NPY Y1 receptor mRNA fluctuated with a clear ovulatory cyclicity, the expression of the receptor gene was acutely increased after the gonadotropin surge, rather than before; we have thus proposed that preovulatory increases in pituitary responsiveness to NPY are manifest by post-receptor events, or by unmasking of cryptic receptors. Further RT-PCR experiments have suggested that the increase in Y1 receptor gene expression following the gonadotropin surge is dependent upon neural signals for the surge, viz. NPY secretion on proestrus. It is proposed that signalling via Y1 receptors is up-regulated prior to the elaboration of the surge, and that the major stimulus for receptor gene expression is occupancy and activation of NPY receptors by the peptide. Rebound receptor gene expression may serve as a compensatory mechansim which replenishes receptor precursers for mobilization during the subsequent ovulatory cycle. A primary role of NPY and its receptors during the ovulatory cycle may be to amplify activity at several levels within the reproductive axis, and thereby ensure efficacious signal transfer between hypothalamus, pituitary, and gonads. The amplification of these events by NPY may function to permit robust hormone secretions under adaptive physiological conditions, viz. those in which acceptable metabolic, feeding, photoperiodic conditions prevail.

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